

Interaction of general adaptation syndrome and myocarditis causing ventricular rupture in a black swan (*Cygnus atratus*): case report

Interação de síndrome geral de adaptação e miocardite causando ruptura ventricular em cisne negro (*Cygnus atratus*): relato de caso

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SUMMARY

Case report about the interaction of chronic myocarditis and General Adaptation Syndrome (“stress”) causing rupture of left ventricular wall on a young, female specie of *Cygnus atratus* (black swan), that came to die at the Country Zoo of Taboão da Serra, in São Paulo, during quarantine, after being taken on a long distance travel, under disfavorable temperature and ventilation conditions.

UNITERMS: Stress; General Adaptation Syndrome; Myocarditis; Swan.

INTRODUCTION

This report describes a young, female species of *Cygnus atratus* that died in the Country Zoo of Taboão da Serra, São Paulo, within the initial twelve hours of quarantine, after being taken on a long distance travel, under disfavorable temperature and ventilation conditions.

The corpse was taken to the Pathology Department of the Faculty of Veterinary Medicine and Animal Science of São Paulo University for necropsy and histological examination. Interaction of stress factors was suggested as the leading cause for the animal's death.

Stress, known as General Adaptation Syndrome^{2,5}, recognized in avian population², involves, firstly, tachycardia and tachypnea, due to the liberation of chemical mediators^{2,5,6,8} and may lead also to the development of hypertension^{1,3,6} and a subsequent rise in the myocardial oxygen consumption¹, which, in the event of an inflammatory process and abrupt rise in the oxygen demand, can lead to local ischemia of the cardiac muscle^{1,3,4,5,6}.

MATERIAL AND METHOD

The corpse underwent conventional necropsy procedures for fowls, and the organs were removed for anatomo-pathological examination. For the histopathological exams, tissue fragments of about one cubic centimeter were removed and immediately fixed in a 10% formaldehyde solution. Fragments were then dehydrated in alcohol solutions, diaphanised in xylol, embedded in paraffin and five mm cuts were stained by the Hematoxylin-Eosine staining method.

RESULTS

The necropsy showed the presence of hemopericardium with approximately 10 ml of clotted blood enveloping the heart, rupture of about 1.5 to 2.0 cm long on the left ventricle, near the apex and transversal to the cardiac fibers, with an adhered blood clot in the region (Fig. 1). Another rupture, with a diameter of about 0.5 cm was present on the ventricular septum, communicating left and right ventricular cavities. Two other ruptures, respectively 0.2 and 0.4 cm long, longitudinal to the cardiac fibers, were present on the right ventricular wall. Congested lungs exhibited reddish, focal areas with diameters



Figure 1

Myocardial rupture on left ventricle (A) associated with local hemorrhage in *Cygnus atratus* (black swan).

as large as 0.5 cm, compatible with a hemorrhagic clinical picture. Other viscera showed no noteworthy anatomic-pathologic alterations.

The histopathologic analysis showed extensive kidney hemorrhage, vacuolar degeneration, necrosis of tubular renal cells, and glomerulonephrosis; small intestines exhibited preserved architecture and mild enteritis, characterized by invasion of mononuclear cells on the lamina propria; congested spleen; the liver had an uncharacterized acinar architecture, due to intense degenerative macrodroplets vacuolar process with necrotic areas and multiple, delimiting foci of mononuclear cell infiltrations; congested brain, with mild hemorrhagic areas; myocardium presented extensive hemorrhagic areas, multi-focal necrosed clotted areas, foci of mononuclear cell infiltrations, specially near the area of larger rupture on the ventricle, generating a clinical picture of chronic myocarditis, and rupture of cardiac fibers in several sections.

DISCUSSION

Apprehensiveness is a physiological stressor that, depending on its intensity, can lead to anxiety, shock or terror³. Characterized as General Adaptation Syndrome^{2,5} - with increases on corticosteroid levels - today stress is already recognized in fowls^{2,8}, though it was applied only to mammals.

On a first moment, the animal reacts presenting tachycardia and tachypnea, due to epinephrin liberation^{2,5,7}. These discharges from the autonomic sympathetic nervous system can lead to hypertension^{1,3}, and to a consequent increase on the oxygen consumption by the myocardium¹.

Following this initial stage - known as alarm reaction^{4,5} - comes a clinical picture characterized by marked plummeting of blood pressure and further tachypnea. This hypotension ends up in causing the animal's death, due to hypovolemic shock^{4,5}.

Main alterations, visible at necropsy were lung edema, cardiac failure, congestion of internal organs and ischemia of

peripheral tissues, skin and muscles, including the myocardium^{1,4,8}. Histopathologic examinations allowed to observe necrosis of skeletal and cardiac muscular masses, dilated hepatic sinusoids, hemorrhages on the small intestines and adrenal cortex, renal tubular dilation and necrosis, and brain hemorrhage^{1,4,5,8}. This clinical picture resembles very much that of the Avian Sudden Death Syndrome⁶.

Some similarities can be found by comparing gross and histopathologic findings of this report with those in the literature, mainly regarding hemorrhages on the myocardium, brain, kidney and liver, and spleen and lung congestion.

However, physiopathology of the General Adaptation Syndrome^{2,5}, or of Avian Sudden Death Syndrome⁶, does not explain the rupture of the ventricular wall in this specimen of *Cygnus atratus*, nor the presence of an inflammatory process in the myocardium.

In regard to this, it's interesting to quote Bogliolo¹, who says "inflammatory processes, after destroying part of the vascular network in the myocardium, can cause local ischemia leading to necrotic clotting of the myocardium, which might occur in those individuals where there is sudden oxygen demand, or in those presenting brusque variations of blood pressure values". These two factors quite likely interacted on this case report^{1,3,4,5}, and this previous alteration of the myocardium would then favor its weakening and give place to the rupture, located near the inflammatory foci, as seen in the report.

Based on those features, we suggest the interaction of physiological stress factors³ - inadequate transportation and intermingling with a unfamiliar environment⁵ - with myocardial deficiency caused by chronic myocarditis as the major factor that led to the necrotic clotting of the myocardium¹, and to the subsequent ventricular rupture, culminating in the hypovolemic shock.

Therefore, this report establishes parameters that differentiate it from Avian Sudden Death Syndrome⁶, initially considered as the cause for the death of this specimen of *Cygnus atratus*; it also allows to consider interaction of General Adaptation Syndrome^{2,5} and Myocarditis¹ as the cause of death.

RESUMO

Relato de caso de interação de miocardite crônica e Síndrome Geral de Adaptação (estresse) causando ruptura da parede ventricular esquerda em uma fêmea jovem da espécie *Cygnus atratus* (cisne-negro), que veio a óbito no Zoológico Municipal de Taboão da Serra, em São Paulo, durante período de quarentena, após longa viagem em condições desfavoráveis de temperatura e ventilação.

UNITERMOS: Estresse; Síndrome Geral de Adaptação; Miocardite; Cisne-Negro.

REFERENCES

- 1- BOGLIOLO, L. **Patologia**. Rio de Janeiro : Guanabara Koogan, 1981. p.47-9.
- 2- COOPER, J.E.; JACKSON, O.F. **Diseases of the reptilia**: London : Academic Press, 1981. p.489-91.
- 3- FOWLER, M.E. **Zoo and wild animal medicine**. Philadelphia : W.B. Saunders, 1986. p.34-5.
- 4- GRINER, L.A. **Pathology of zoo animals**. San Diego : Zoological Society of San Diego, 1983. 608p.
- 5- HOFF, G.L.; DAVIS, J.W. **Noninfectious diseases of wildlife**. Iowa : The State University Press, 1982. p.58-73.
- 6- OLKOWSKI, A.A.; CLASSEN, H.L. Sudden death syndrome in broiler chicken: a review. **Poultry and Avian Biology Reviews**, v.6, n.2, p.95-105, 1995.
- 7- RANDALL, C.J. **A colour atlas of disease of the domestic fowl and turkey**. England : Wolf Medical, 1985. p.81-7.
- 8- SEAL, U.S.; BUSH, M. Capture and chemical immobilization of cervids. In: **BIOLOGY AND MANAGEMENT OF THE CERVIDAE**. Washington, DC : Smithsonian Institution Press, 1982. p.480-97. (Research Symposia of the National Zoological Park)

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